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### The Demon Drink

Mark Nelson

*University of Wollongong*, mnelson@uow.edu.au

Peter Hagedoorn

*University of Wollongong*

Annette L. Worthy

*University of Wollongong*, annie@uow.edu.au

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# The Demon Drink

## Abstract

We provide a qualitative analysis of a system of nonlinear differential equations that model the spread of alcoholism through a population. Alcoholism is viewed as an infectious disease and the model treats it within a sir framework. The model exhibits two generic types of steady-state diagram. The first of these is qualitatively the same as the steady-state diagram in the standard sir model. The second exhibits a backwards transcritical bifurcation. As a consequence of this, there is a region of bistability in which a population of problem drinkers can be sustained, even when the reproduction number is less than one. We obtain a succinct formula for this scenario when the transition between these two cases occurs.

## Disciplines

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## The demon drink

MARK IAN NELSON<sup>✉ 1</sup>, PETER HAGEDOORN<sup>2</sup> and ANNETTE WORTHY<sup>3</sup>

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### Abstract

We provide a qualitative analysis of a system of non-linear differential equations that model the spread of alcoholism through a population. Alcoholism is viewed as an infectious disease and the model treats it within a SIR framework. The model exhibits two generic types of steady-state diagram. The first of these is qualitatively the same as the steady-state diagram in the standard SIR model. The second exhibits a backwards transcritical bifurcation. As a consequence of this there is a region of bistability in which a population of problem drinkers can be sustained even when the reproduction number is less than one. We obtain a succinct formula for when the transition between these two cases occurs.

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## 1. Introduction

The field of mathematical epidemiology models the spread of diseases through a population. The most common approach to model an endemic disease is to use the SIR framework in which individuals in a population are classed as being in one of three compartments: susceptible, that is yet to be infected, infectious or recovered with immunity. This framework can be extended to include additional classes, such as an exposed class for individuals who have caught the disease but are not yet infectious, or contracted by removing the recovered class; in this case recovered individuals do not acquire immunity and return into the susceptible class.

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<sup>1</sup>School of Mathematics and Applied Statistics, University of Wollongong, Wollongong, NSW 2522, AUSTRALIA; e-mail: [nelsonm@member.ams.org](mailto:nelsonm@member.ams.org), [mnelson@uow.edu.au](mailto:mnelson@uow.edu.au) <http://orcid.org/ORCID#0000000239645928>.

<sup>2</sup>School of Mathematics and Applied Statistics, University of Wollongong, Wollongong, NSW 2522, AUSTRALIA; e-mail: [annie@uow.edu.au](mailto:annie@uow.edu.au).

<sup>3</sup>School of Mathematics and Applied Statistics, University of Wollongong, Wollongong, NSW 2522, AUSTRALIA; e-mail: [ph247@uowmail.edu.au](mailto:ph247@uowmail.edu.au).

The field of mathematical epidemiology was brought to the attention of a wider audience by Anderson and May [1]. As befits a mature discipline there are now textbooks devoted to mathematical epidemiology [3, 28, 33], lecture notes for graduate students [4] and an acknowledged modern classic [7].

In recent years the SIR framework has been applied to study the dynamics of social and behavioural processes such as drug use [27, 35], eating disorders [11], obesity [14, 20], smoking [17, 32] and alcoholism. The SIR analogy can be employed when the problem ‘state’, i.e. the infection, can be viewed as occurring as the result of frequent or intense social interactions between individuals in different compartments. The analogy with the spread of diseases is clear. The application of the SIR framework to problems in sociology that are driven by peer pressure is reviewed, through the prism of projects developed at the Mathematical and Theoretical Biology Institute, by Kribs-Zaleta [23].

Alcohol is the most commonly used addictive substance throughout the world leading to approximately 3.3 million deaths per year, both by alcohol-induced diseases and through risky behaviour induced by over-consumption of alcohol [21]. Individuals who consume alcohol at a young age are at a greater risk of developing heavier drinking patterns throughout adolescence and adulthood, leading to a greater chance of developing adverse physical and mental health conditions [15].

Although Australia has never seen a nation-wide prohibition on the sale of alcohol, the only successful armed takeover of an Australian government was the Rum Rebellion of 1808. Within Australian popular culture notable events connected to alcohol consumption include cricketer David Boon putting away a legendary 52 cans of beer on the flight from Sydney to London prior to the 1989 Ashes series and future Prime Minister Bob Hawke downing a yard of ale in 11 seconds whilst a student at the University of Oxford in the early to mid 1950s.

Australian politicians penchant to use beer drinking to connect to voters has been documented in the popular press [19]. With regard to this it is interesting to observe that “Australians appear to be drinking less alcohol now than at any time in the past 50 years” [13]. Furthermore, the proportion of alcohol consumed in the form of wine has increased from 12% to 38% over the past fifty years, whilst the proportion of alcohol consumed in the form of beer has reduced significantly from approximately 75% to 41% [13]. One is forced to wonder whether a politician’s predilection for a beer is a sign that they are out-of-touch with the general public. Alternatively, perhaps they have identified that floating voters are more likely to drink beer.

We present an analysis of a model for the spread of alcoholism through a population that was derived by Sanchez *et al* [31]. We analyse this model in more detail than was reported previously and obtain compact explicit representations in parameter space delineating regions of different generic behaviour.

An interesting issue related to the spread of ‘social diseases’ is the role played by advertising campaigns in encouraging or discouraging certain behaviour. How do ‘positive’ and/or ‘negative’ media campaigns influence the long-term behaviour of a population? In latter work we intend to extend the model of Sanchez *et al*

[31] to include the impact of pro-alcohol and anti-alcohol media campaigns upon the behaviour of a population [12]. The thorough re-analysis of Sanchez's model presented here therefore provides the necessary foundations for a study of these media effects.

## 2. Model equations

The underlying model is the standard SIR (Susceptible, Infected, Recovered) model for the spread of a disease through a population. Thus the population is divided into three compartments and it is assumed that all individuals within these compartments, regardless of age or sex, behave in the same manner. The compartments represent: occasional and moderate drinkers ( $S$ ), problem drinkers ( $I$ ) and temporarily recovered drinkers ( $R$ ). It is assumed that moderate drinkers become problem drinkers through their interaction with problem drinkers. Consequently problem drinkers are viewed as infecting social drinkers. As noted in §§ 3.1 the relapse of individuals from the class of recovers to the class of problem drinking is assumed to occur through peer pressure.

**2.1. Dimensional equations** The model equations are given by [31]

$$\frac{dS}{dt} = \mu N - \frac{\beta}{N} S I - \mu S, \quad (1)$$

$$\frac{dI}{dt} = \frac{\beta}{N} S I - (\gamma + \mu) I + \frac{p}{N} R I, \quad (2)$$

$$\frac{dR}{dt} = \gamma I - \mu R - \frac{p}{N} R I, \quad (3)$$

$$N = S + I + R. \quad (4)$$

In these equations:  $I$  is the number of problem drinkers,  $N$  is the total number of drinkers,  $R$  is the number of recovered drinkers,  $S$  is the number of occasional drinkers,  $p$  is the per-person relapse rate (units,  $|t|^{-1}$ ), a measure of the average number of effective interactions between problem and recovered drinkers per unit time,  $t$  is time (units,  $|t|$ ),  $\beta$  is the transmission rate (units,  $|t|^{-1}$ ), a measure of the average number of effective interactions between social and problem drinkers per unit time, and  $\gamma$  is the per-capita removal rate from the problem drinking class to the recovered drinking class (units,  $|t|^{-1}$ ).

In equation (1) the parameter  $\mu$  (units,  $|t|^{-1}$ ), is the per-capita rate at which new recruits join the drinking population. In equations (2) & (3) it represents the per-capita departure rate of individuals from the drinking population. These processes are assumed equal to ensure that the total population size is constant.

Note that from equations (1)–(3) the total population size is constant,

$$N(t) = S(0) + I(0) + R(0), \quad (5)$$

where the quantities on the right-hand side are the number of individuals in the three classes at  $t = 0$ .

**2.2. Semi-scaled equations** The compartments are scaled so that each represents the proportion of the population in it by setting  $s = S/N$ ,  $i = I/N$  and  $r = R/N$ . This leads to the set of equations

$$\frac{ds}{dt} = \mu - \beta si - \mu s, \quad (6)$$

$$\frac{di}{dt} = \beta si - (\gamma + \mu)i + pri, \quad (7)$$

$$\frac{dr}{dt} = \gamma i - \mu r - pri. \quad (8)$$

It follows from equation (5) that the solution of this system satisfies

$$s + i + r = 1. \quad (9)$$

Using the conservation relationship (9) the system of three differential equations, equations (6)–(8), reduces to the planar system

$$\frac{ds}{dt} = \mu - \beta si - \mu s, \quad (10)$$

$$\frac{di}{dt} = \beta si - (\gamma + \mu)i + pi(1 - s - i). \quad (11)$$

The Jacobian matrix for this system is given by.

$$J(s, i) = \begin{pmatrix} -\beta i - \mu & -\beta s \\ (\beta - p)i & \beta s - (\gamma + \mu) + p - ps - 2pi \end{pmatrix}. \quad (12)$$

### 3. Alcoholism and Epidemiology

In recent years a number of authors have investigated the spread of alcoholism through a population through the use of compartmental models [2, 5, 6, 16, 18, 24, 25, 26, 31, 34]. These models are based on the classic SIR model. The primary distinguishing feature between the standard SIR model and models for alcoholism is that in the former the recovered population ( $R$ ) acquire life-long immunity to the disease. This is not the case for alcoholism as recovered drinkers may, and usually do, relapse back into the class of ‘infectives’ (problem drinkers) ( $I$ ).

We differentiate models for the spread of alcoholism on whether relapse is due to ‘peer pressure’, discussed in section 3.1, or due to the individual, discussed in section 3.2. Models in which relapse is not included are discussed in section 3.3.

Unless otherwise specified the models discussed assume that alcoholism does not increase the natural removal rate of individuals from compartments. Consequently the total population size remains constant. This assumption is justified for models which consider the spread of drinking over population cohorts with a narrow spread of age, such as students on university campuses or, more generally, youths.

**3.1. Models with relapse due to ‘peer pressure’** The first paper to apply the SIR paradigm to modelling the spread of alcoholism was published by Sanchez *et al* [31]. These authors studied drinking in a college population. It is assumed that problem drinkers ( $I$ ) can not relapse back into the social drinking class ( $S$ ) and that recovered drinkers ( $R$ ) can only relapse back into the problem drinking class; i.e. they can not become social drinkers.

To incorporate peer pressure the relapse rate ( $\mathcal{R}$ ) is modelled by a ‘bimolecular’ rate term that is first order in both the number of recovered individuals and the number of infectives:

$$\mathcal{R} = \frac{p}{N}RI.$$

It is shown that in some circumstances the effect of peer pressure is to allow problem drinkers to be endemic in a population even when the basic reproduction number is less than one. This phenomenon occurs through a so-called ‘backwards bifurcation’ [30].

Cintrón-Arias *et al* [6] adapted the deterministic modelling framework of Sanchez *et al* [31] to two stochastic settings. In the first setting the stochastic analog (continuous-time Markov chain) of the deterministic model was derived and simulated. They showed that stochastic fluctuations can allow a community of problem drinkers to be sustained under conditions in which it would become extinct in the deterministic model.

In the second setting the authors investigated the effect of community structure upon the spread of problem drinkers. In the absence of relapse it was found that community structure affects the average size of the problem drinking class during the drinking wave. However, for larger values of the relapse parameter the community structure has no impact on the prevalence of the problem drinking class.

**3.2. Models with relapse due to the individual** The papers discussed in this section model relapse from the ‘recovered’ class ( $R$ ) to the class of problem drinkers ( $I$ ) by

$$\mathcal{R} = p'R.$$

When this functional form is used relapse is ascribed to an individuals failure to control alcohol withdrawal symptoms rather than peer pressure.

Bhunu [2] investigated a model containing four compartments: non-drinkers, social drinkers, alcoholics and recovered. A term representing alcohol induced death is included. Thus the total population size is a function of time. There are two steady-state solutions: an alcohol free equilibrium, in which all individuals are non-drinkers, which is locally asymptotically stable when the basic reproduction number is less than one ( $R_0 < 1$ ), and an endemic equilibrium, in which a positive fraction of the population are social drinkers and alcoholics, which is locally asymptotically stable when the reproduction number is slightly greater than one ( $R_0 = 1 + \epsilon$ ). Model parameters represent typical values for developing countries. A combination of analytical and numerical methods are used to investigate whether it is more effective to encourage social drinkers or alcoholics to quit alcohol consumption.

Huo and Song [16] investigated an SIR model for binge drinking containing four compartments. The 'I' compartment, representing heavy drinkers, is split into two groups, differentiating between individuals that admit/do not admit that they have a problem. The behavior of this model is essentially the same as that of the classic SIR model: either the disease-free state or the endemic state is globally asymptotically stable depending upon if the basic reproduction number is less than or greater than one. The disease-free state corresponds to a scenario in which the population consists of either non-drinkers or individuals who drink in moderation.

Huo and Wang [18] considered a four compartment model in which the class of individuals who drink heavily and recognise that they have a problem is replaced by a class of non-drinkers. They investigated the effect that anti-drinking media campaigns have on drinking behaviour. They did this by adding a fifth compartment which represents the cumulative density of awareness programs. The media campaign is assumed to convert social drinkers into non-drinkers. There are only two steady-state solutions: a disease-free equilibrium, which is globally asymptotically stable for  $R_0 \leq 1$ , and an endemic equilibrium, which is conjectured to be globally asymptotically stable for  $R_0 > 1$ . The disease-free state corresponds to a scenario in which the population consists of either non-drinkers or individuals who drink in moderation.

Three and four-component models for binge drinking were developed and analysed by Mulone & Straughan [26]. The former differs from that of Sanchez *et al* [31] only in the functionality of the relapse rate. In the latter the class of heavy drinkers ( $I$ ) is split into two: those who admit that they have a problem and those that do not admit that they have a problem.

For both models there are only two steady-state solutions: a disease-free equilibrium, in which the population consists of occasional and moderate drinkers, which is locally asymptotically stable for  $R_0 < 1$ , and an endemic equilibrium, in which there is a positive fraction of problem drinkers, which is locally asymptotically stable for  $R_0 > 1$ . A notable feature of this paper is the attempt to estimate realistic parameter values for binge drinkers in the north east of the UK.

Walters *et al* [34] extended the three-compartment model of Mulone & Straughan [26] by allowing some of the individuals in the recovered population to move into the social drinker class. There are only two steady-state solutions: a disease-free equilibrium, which is locally asymptotically stable for  $R_0 < 1$ , and an endemic equilibrium, which is locally asymptotically stable for  $R_0 > 1$ . Significant features of this paper include a sensitivity analysis, to determine which model parameter has the greatest effect on the value of  $R_0$ , and the estimation of parameter values based on information about binge drinking in England.

The model of Walters *et al* [34] has been extended by Buonomo & Lacitignola [5]. The differences in the model are threefold. Firstly, it is assumed that the population is recruited at a constant rate irrespective of the size of the population. Secondly, it is assumed that alcoholics have a higher mortality rate. The consequence of these two assumptions is that, unlike in [34], the total population size is no longer constant.

The final, and most significant, difference is that the rate at which susceptibles



becomes alcoholics ( $B(S, I)$ ) is modelled by a function originating in management science [9]

$$B(S, I) = \beta SI(1 + \alpha I).$$

Here the term  $\alpha I$  represents an increase in the conversion rate due to additional social pressure that is characterised as imitation: ‘if everyone else is doing it, shouldn’t I do it?’. It is shown that, as in the original model of Sanchez *et al* [31], that a backward bifurcation can occur.

Relapse due to the individual can occur in modelling the spread of diseases. For example, individuals may lose their immunity to a disease and relapse into the infectives group. Other possibilities in the context of diseases include infectives and/or recovered individuals relapsing into the pool of susceptibles. The impact of these types of relapse for the spread of diseases and their consequences for vaccination campaigns have been investigated by Gomes *et al* [10].

**3.3. Models with no relapse** Manthey *et al* [24] developed a model to study the drinking of college students. The compartments in the model were ‘non-drinkers’, social drinkers and problem drinkers; a recovered compartment is not included and consequently a relapse function is not required. The model has three steady-state solutions: alcohol-free (the population only consists of non-drinkers), problem drinking-free (the population consists of non-drinkers and social drinkers), and endemic (the population contains problem drinkers). As in the study of Sanchez *et al* [31] it was found that reproduction numbers on their own are not sufficient to predict whether drinking behaviour is endemic in the population. It was shown that alcohol abuse can be reduced by minimizing the ability of problem drinkers to recruit nondrinkers.

Mubayi *et al* [25] investigated a model for drinking within the US college environment. A unique feature of this model is that it includes two environments: a low risk and a heavy risk environment. Two compartments are associated with each of the two environments. For the low risk environment these are social drinkers and low-risk moderate drinkers. For the high risk environment they are high-risk moderate drinkers and heavy drinkers. Drinkers in the low-risk moderate class may move into the high-risk moderate class and vice-versa. As in Manthey *et al* [24] a recovered compartment is not included and consequently a relapse function is not required.

There are only two steady-state solutions: a disease-free equilibrium, in which the population consists of light or occasional drinkers, which is globally asymptotically stable for  $R_0 \leq 1$ , and an endemic equilibrium, in which the populations also contains moderate and heavy drinkers, which locally asymptotically stable when the reproduction number is slightly greater than one ( $R_0 = 1 + \epsilon$ ). Parameter estimates are based upon regional national college data.

Mubayi *et al* [25] investigate how the relative residence times of moderate drinkers between the low-risk and high-risk environments influences the persistence of heavy drinking. If this distribution occurs randomly, then the proportion of heavy drinkers is likely to be higher than expected.

## 4. Results

In §§ 4.1 we state some global results for the system (6)–(8) and for the planar system (10) & (11).

In §§ 4.2 we find the steady-state solutions of the planar system (10) & (11) and state the conditions under which they are physically meaningful. In §§ 4.3 we determine the stability of these solutions. Steady-state diagrams are presented for the types of (physically meaningful) behaviour exhibited by the model.

Two important parameters in the analysis of the model equations are

$$R_0 = \frac{\beta}{\mu + \gamma}. \quad (13)$$

$$R = \frac{\beta}{\mu}, \quad (14)$$

(Note that in [31] the notation  $R_0$  and  $R_\gamma$  is used instead of  $R$  and  $R_0$  respectively).

The first parameter,  $R_0$ , is the basic reproduction number, i.e. it is the number of secondary infections caused by a single alcoholic in a population of susceptible individuals. The second parameter,  $R$ , is the number of infections caused by a single alcoholic in a population of susceptible individuals in the absence of recovery. We take the former as the primary bifurcation parameter and investigate the behaviour of the model as a function of  $R_0$  at a fixed value of  $R$ . Thus we are effectively taking the rate at which infected drinkers seek treatment ( $\gamma$ ) as the primary bifurcation parameter. Increasing the value of this parameter *decreases* the value of the basic reproduction number due to recovery of infected individuals.

Note that the number  $R$  is strictly greater than the basic reproduction number of infectives seeking treatment ( $R_0$ ) except if the recovery rate ( $\gamma$ ) is zero.

**4.1. Global results** In this section we state some global results about the behaviour of the system (1)–(3) and the planar system (10) & (11).

Firstly, the region  $\mathcal{I}$  defined by

$$\begin{aligned} 0 &\leq s, \\ 0 &\leq i, \\ 0 &\leq r, \\ s + i + r &= 1 \end{aligned}$$

is positively invariant.

Secondly, there are no limit-cycles in the planar system (10) & (11).

Thirdly, if  $R < 1$  it is not possible to establish a culture of problem drinkers in the community, i.e.

$$\lim_{t \rightarrow \infty} i(t) = 0.$$

These results are established in §§ A.

**4.2. Steady State Solutions** In this section we find the steady state solutions of the planar system (10)-(11). The planar system has two steady-state solution branches.

The disease-free steady-state

$$(s, i) = (1, 0). \quad (15)$$

The endemic steady-state

$$(s, i) = (\hat{s}, \hat{i}), \quad (16)$$

$$\hat{s} = \frac{p}{\beta - p} \cdot \hat{i} + \frac{1}{\beta - p} (\gamma + \mu - p), \quad (17)$$

where  $\hat{i}$  is a root of the quadratic equation

$$\mathcal{G}(\hat{i}) = \hat{i}^2 - \left(1 - \frac{1}{R} - \frac{1}{R_0} \frac{\beta}{p}\right) \hat{i} + \frac{\mu}{p} \left(\frac{1}{R_0} - 1\right) = 0. \quad (18)$$

The disease-free steady-state represents the scenario in which the population contains no problem drinkers. When the endemic steady state is physically meaningful it represents alcoholism, in the form of problem drinkers, becoming established within the population.

In §§ B we determine the conditions under which equation (18) has positive roots. The first requirement is that

$$R = \frac{\beta}{\mu} > 1. \quad (19)$$

(This makes sense as from §§ 4.1 we know that problem drinkers are eradicated from the population when  $0 \leq R < 1$ ).

If

$$p \leq p_{\text{cr}} = \frac{R}{R-1} \cdot \beta$$

then equation (18) has no positive solutions for  $0 < R_0 \leq 1$  and one positive solution for  $1 < R_0 < R$ .

If

$$p > p_{\text{cr}}$$

then equation (18) has no positive solutions for  $0 < R_0 < R_+$ , one positive solution at  $R_0 = R_+$ , two positive solutions for  $R_+ < R_0 < 1$  and one positive solutions for  $1 \leq R_0 < R$ . The value  $R_+$  is defined by equation (41).

Note that an alternative expression for the steady-state solution for the fraction of social drinkers can be obtained from equation (10)

$$\hat{s} = \frac{\mu}{\beta \hat{i} + \mu} = \frac{1}{R \hat{i} + 1}.$$

This formulation shows that if the value for the fraction of problem drinkers is physically meaningful ( $\hat{i} > 0$ ) then so is the corresponding fraction of social drinkers.

**4.3. Stability** In §§ 4.3.1 we determine the stability of the disease-free steady-state. In §§ 4.3.2 we determine the stability of the endemic steady-state solution(s), provided that it is physically meaningful. For the latter, three cases are considered depending upon the value of the relapse rate ( $p$ ).

*4.3.1. The disease-free steady-state* For the disease-free steady state solution  $(s, i) = (1, 0)$  the Jacobian matrix (12) becomes

$$J(1, 0) = \begin{pmatrix} -\mu & -\beta \\ 0 & \beta - (\gamma + \mu) \end{pmatrix}.$$

The eigenvalues of the Jacobian matrix are

$$\begin{aligned} \lambda_1 &= -\mu < 0, \\ \lambda_2 &= \beta - (\gamma + \mu), \\ &= \beta \cdot \frac{R_0 - 1}{R_0}. \end{aligned}$$

Thus the disease-free steady-state solution is locally stable (unstable) if the basic reproduction number ( $R_0$ ) is less than (greater than) one. We shall see latter that the condition  $R_0 < 1$  is not always sufficient to ensure eradication of problem drinkers from the population.

*4.3.2. The endemic steady-state(s)* The entry in the Jacobian matrix at position  $J_{(2,2)}$  can be simplified for the endemic steady-state using the steady-state expression for the susceptible fraction (17). Dropping the hat notation for convenience the Jacobian matrix becomes

$$J(s, i) = \begin{pmatrix} -\beta i - \mu & -\beta s \\ (\beta - p) i & -p i \end{pmatrix}.$$

We have

$$\text{trace} J = -(\beta + p) i - \mu, \quad (20)$$

$$\det J = i [2\beta p i + p(\mu - \beta) + \beta(\mu + \gamma)], \quad (21)$$

$$= \beta i \left[ 2p i + p \cdot \frac{1 - R}{R} + \frac{\beta}{R_0} \right]. \quad (22)$$

For physically meaningful solutions we have  $\text{trace} J < 0$ , thus the condition for stability is  $\det J > 0$ . We consider the three cases:  $p < p_{cr}$ ,  $p = p_{cr}$  and  $p > p_{cr}$ .

*4.3.3. Case 1:  $p < p_{cr}$*  We know from §§ 4.2 that when  $p < p_{cr}$  that the endemic steady-state solution is not physically meaningful for  $R_0 \leq 1$ . For  $R_0 > 1$  there is a unique physically meaningful endemic steady-state. From equation (18) this solution

is given by

$$i_+ = \frac{1}{2} \left\{ \left[ \left( \frac{R-1}{R} \right) - \frac{1}{R_0} \frac{\beta}{p} \right] + \sqrt{\left[ \left( \frac{R-1}{R} \right) - \frac{1}{R_0} \frac{\beta}{p} \right]^2 - \frac{4\mu}{p} \left( \frac{1-R_0}{R_0} \right)} \right\}, \quad (23)$$

$$> \frac{1}{2} \left[ \left( \frac{R-1}{R} \right) - \frac{1}{R_0} \frac{\beta}{p} \right]. \quad (24)$$

It follows that the determinant (22) is positive for  $R_0 > 1$ . Hence this solution is stable.

The steady-state diagram for this case is shown in Figure 1. As the transcritical bifurcation occurs in the ‘forwards’ direction, the condition  $R_0 \leq 1$  ensures that problem drinkers are eradicated from the population.

When  $R_0 \leq 1$  the disease-free steady-state solution is the only steady-state solution. From §§ 4.1 we know that the region

$$\begin{aligned} 0 &\leq s, \\ 0 &\leq i \leq 1 - s, \end{aligned}$$

is both positively invariant and contains no periodic orbits. It follows from the generalized Poincaré-Bendixon Theorem [29, page 243] that the disease-free steady-state is globally stable.

When  $R_0 > 1$  there are two steady-state solutions. The disease-free steady-state solution is locally unstable whilst the endemic steady-state solution is locally stable. The disease-free steady-state has a one-dimensional stable manifold which is the line  $i = 0$ . It follows from the generalized Poincaré-Bendixon Theorem [29, page 243] that the endemic steady-state solution is globally stable for all initial conditions with  $i(0) > 0$ .

4.3.4. *Case 2:  $p = p_{cr}$*  For this case the determinant (22) simplifies to

$$\det J = \beta i \left[ 2pi + \beta \left( \frac{1}{R_0} - 1 \right) \right]. \quad (25)$$

We know from §§ 4.2 that when  $p = p_{cr}$  that the endemic steady-state solution is not physically meaningful for  $R_0 \leq 1$ . For  $R_0 > 1$  there is a unique physically meaningful endemic steady-state. It immediately follows from the argument observed in §§ (4.3.3) that for this solution ( $i_+$ ) we have  $\det J > 0$ .

The steady-state diagram for this case is shown in Figure 2. As in Figure 1 the condition  $R_0 \leq 1$  ensures that problem drinkers are eradicated from the population.

The generalized Poincaré-Bendixon Theorem can again be applied to deduce that the disease-free steady-state is globally stable when it is locally stable and that the endemic steady-state is globally stable when it is locally stable, excepting initial conditions starting on the line  $i = 0$  which converge to the disease-free steady-state.

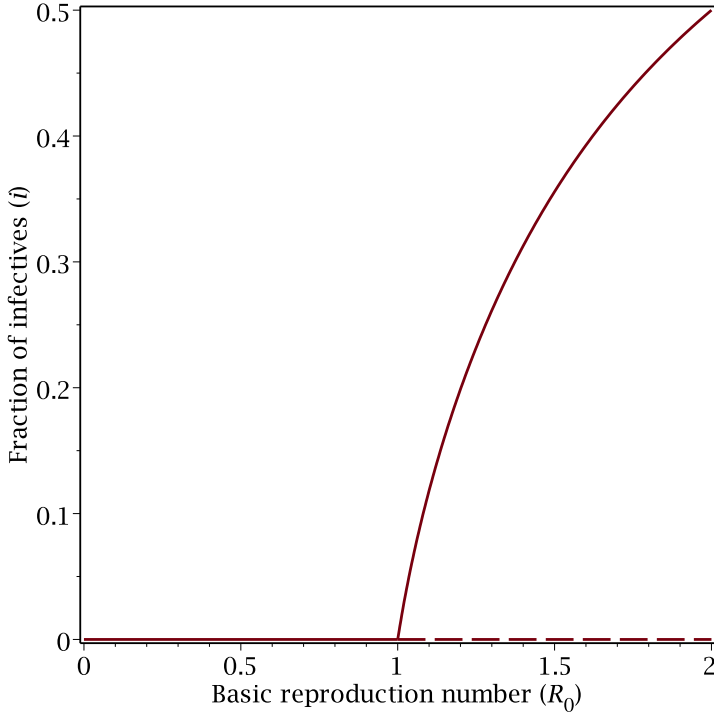


FIGURE 1. Steady-state diagram for the case when the relapse rate is less than the critical relapse rate ( $p < p_{cr}$ ). There is a transcritical bifurcation at the point  $(R_0, i) = (1, 0)$ . The solid and dashed lines indicate of stable and unstable steady-state solutions respectively. Parameter values  $\beta = 0.8$ ,  $\mu = 0.4$ ,  $R = 2$ ,  $p = 1.1$ ,  $p_{cr} = 1.6$ .

4.3.5. *Case 3:  $p > p_{cr} = \frac{\beta^2}{\beta - \mu}$*  We know from §§ 4.2 that when  $p > p_{cr}$  that the endemic steady-state equation (18) has two positive solutions ( $i_-$  and  $i_+$ ) over the range  $R_+ < R_0 < 1$  and one positive solution ( $i_+$ ) for  $1 \leq R_0 < R$ . It immediately follows from the argument observed in §§ (4.3.3) that for the steady-state solution  $i_+$  we have  $\det J > 0$ . Hence this steady-state solution is stable.

For the (positive) solution  $i_-$  we have

$$i_- = \frac{1}{2} \left\{ \left[ \left( \frac{R-1}{R} \right) - \frac{1}{R_0} \frac{\beta}{p} \right] - \sqrt{\left[ \left( \frac{R-1}{R} \right) - \frac{1}{R_0} \frac{\beta}{p} \right]^2 - \frac{4\mu}{p} \left( \frac{1-R_0}{R_0} \right)} \right\}, \quad (26)$$

$$< \frac{1}{2} \left[ \left( \frac{R-1}{R} \right) - \frac{1}{R_0} \frac{\beta}{p} \right]. \quad (27)$$

It follows that when the solution  $i_-$  is physically meaningful ( $i_- > 0$ ) that the determinant (22) is negative. Hence this solution is unstable.

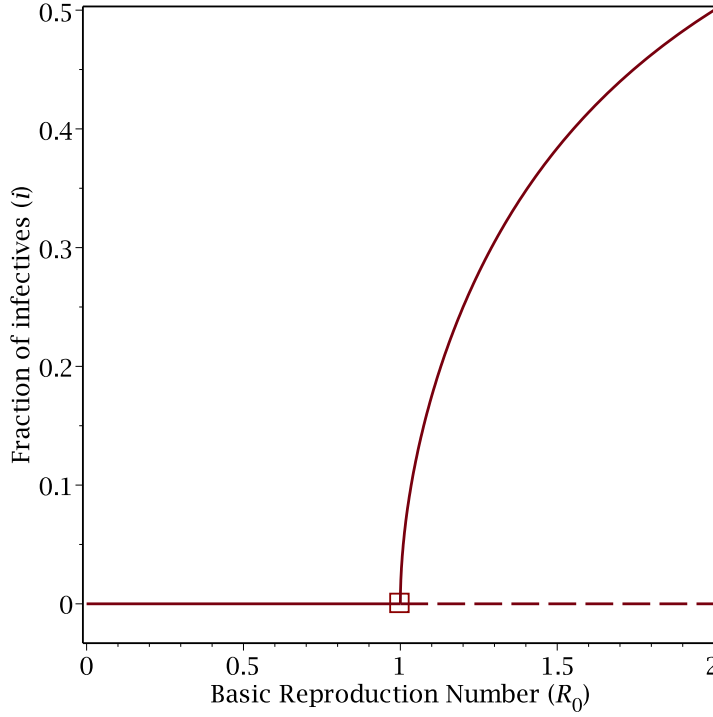


FIGURE 2. Steady-state diagram for the case when the relapse rate is equal to the critical relapse rate ( $p = p_{\text{cr}}$ ). There is a pitchfork bifurcation at the point  $(R_0, i) = (1, 0)$ . The solid and dashed lines indicate of stable and unstable steady-state solutions respectively. Parameter values  $\beta = 0.8$ ,  $\mu = 0.4$ ,  $R = 2$ ,  $p = p_{\text{cr}} = 1.6$ .

The steady-state diagram for this case is shown in Figure 3. The practical consequence of the backwards bifurcation at  $R_0 = 1$  is that the condition  $R_0 \leq 1$  is no longer a sufficient condition to eradicate problem drinkers from the population. To ensure removal of all problem drinkers we require  $R_0 < R_+$ : the steady-state diagram exhibits bistability over the region  $R_+ \leq R_0 \leq 1$ .

As in §§ 4.3.3 & 4.3.4 the generalized Poincaré-Bendixon Theorem can be used to make deductions regarding the global stability of the disease-free and endemic steady-state solutions. However, this theorem is only applied in the ‘unstable’ parameter regions  $0 < R_0 < R_+$  and  $R_0 > 1$ .

This is the most ‘interesting’ of the three cases, since it shows that at sufficiently high relapse rates ( $p > p_{\text{cr}}$ ) a population of problem drinkers may be maintained in a population even when the reproduction number ( $R_0$ ) is reduced below one.

Sanchez *et al* [31, Figure 16.4] provide the following typical values for the backwards bifurcation case:  $\mu = 5.48 \times 10^{-5} \text{ t}^{-1}$ ,  $p = 0.21 \text{ t}^{-1}$  and  $\gamma = 0.2 \text{ t}^{-1}$ . In this case the primary bifurcation parameter is the transmission rate  $\beta$ . We find that there

is a backwards transcritical bifurcation at  $\beta = 0.2001 \text{ t}^{-1}$ , corresponding to  $R_0 = 1$  and that the limit-point bifurcation occurs at  $\beta = 0.06510 \text{ t}^{-1}$ . This corresponds to a value  $R_0 = R_+ = 0.325$ .

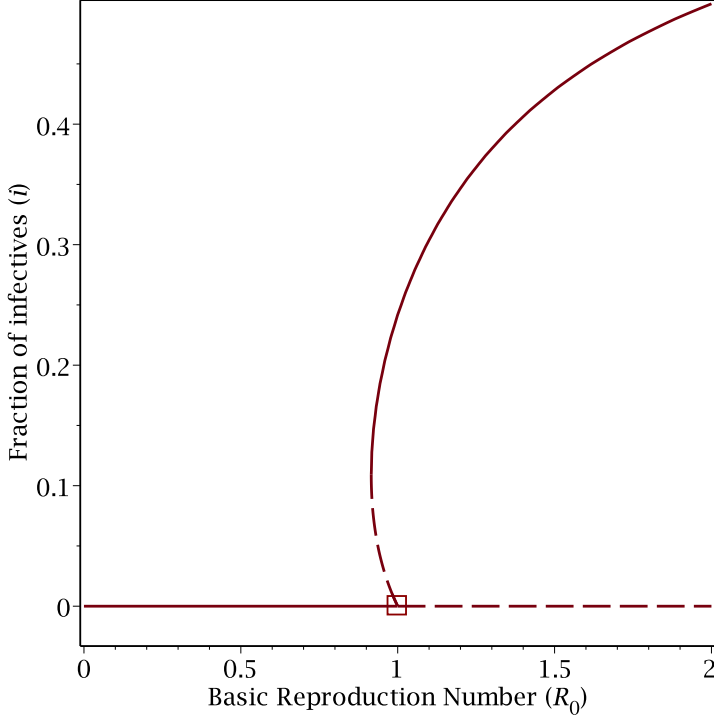


FIGURE 3. Steady-state diagram for the case when the relapse rate is greater than the critical relapse rate ( $p > p_{cr}$ ). There is a transcritical bifurcation at the point  $(R_0, i) = (1, 0)$  and a limit-point bifurcation at the point  $(R_+, \hat{i}(R_+))$ . The solid and dashed lines indicate of stable and unstable steady-state solutions respectively. Parameter values  $\beta = 0.8$ ,  $\mu = 0.4$ ,  $R = 2$ ,  $p = 3.1$ ,  $p_{cr} = 1.6$ .

## 5. Conclusions

In this paper we have re-analysed a model for the spread of alcoholism through a population that was proposed by Sanchez *et al* [31]. Amongst other results we have shown that periodic solutions are impossible and we have determined the stability of the steady-state solutions as a function of the primary bifurcation parameter.

Sanchez *et al* [31] showed, provided  $R > 1$ , that their model exhibits two generic types of steady-state diagrams. In the first type there is a ‘forwards’ transcritical bifurcation, at  $R_0 = 1$ . In this case the steady-state diagram, shown in Figure 1, is qualitatively the same as that in the standard SIR model. In the second type there is a ‘backwards’ transcritical bifurcation, at  $R_0 = 1$ , and a limit-point bifurcation, at



$R_0 = R_+ < 1$ . This leads to a region of bistability,  $R_+ < R_0 < 1$ , in which a non-zero population of problem drinkers can be maintained, even though the basic reproduction number is less than one. This steady-state diagram is shown in Figure 3.

We have obtained a simple criterion that identifies when each region occurs. If the per-person relapse rate is sufficiently low (high) then the first (second) type of behaviour occurs. The critical value at which the transition occurs between the two types is given by

$$p = p_{\text{cr}} = \frac{R}{R-1} \cdot \beta.$$

Note that, in particular, bistability is impossible if the per-person relapse rate ( $p$ ) is lower than the transmission rate ( $\beta$ ).

The thorough re-analysis of the model due to Sanchez *et al* [31] presented here lays the foundations for an investigation into the role played by the media in encouraging/discouraging binge drinking [12]. These results will be presented at a latter date.

### Acknowledgements

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## A. Global results

**A.1. Solutions are non-negative** The initial conditions of the system are non-negative. We show that the solution components of equations (6)–8 are non-negative, i.e.

$$\begin{aligned} 0 &\leq s(t), \\ 0 &\leq i(t), \\ 0 &\leq r(t). \end{aligned}$$

To do this we consider the derivative of each solution component when the latter is equal to zero.

For the social drinking class we have from equation (6) that

$$\left. \frac{ds}{dt} \right|_{s=0} = \mu > 0. \quad (28)$$

Therefore, the fraction of social drinkers is always non-negative.

For the problem drinking class we have from equation (7) that

$$\left. \frac{di}{dt} \right|_{i=0} = 0. \quad (29)$$

This shows that the line  $i = 0$  is both positively and negatively invariant. Therefore, the fraction of problem drinkers is always non-negative.

For the temporarily recovered drinking class we have from equation (8) that

$$\left. \frac{dr}{dt} \right|_{r=0} = \gamma i \geq 0 \quad \text{as } i \geq 0. \quad (30)$$

Therefore, the fraction of temporarily recovered drinkers is always non-negative.

**A.2. There are no limit cycles** To show the non-existence of a periodic solution we use Dulac's criteria.

**THEOREM 1 (Dulac's Test [8, 22]).** *Consider the system*

$$\begin{aligned} \frac{dx}{dt} &= f(x, y), \\ \frac{dy}{dt} &= g(x, y). \end{aligned}$$

*Let  $\mathcal{D}$  be a simply connected region in  $\mathbb{R}^2$  and let  $\{f(x, y), g(x, y)\} \in C^1(\mathcal{D})$ . If there exists a function  $\rho \in C^1(\mathcal{D})$  such that*

$$\frac{\partial(\rho f)}{\partial x} + \frac{\partial(\rho g)}{\partial y}$$

*is not identically zero and does not change sign in  $\mathcal{D}$ , then this system does not have any closed paths lying entirely in  $\mathcal{D}$ .*

We use the test function  $\rho = 1/i$ . To see that this test function is acceptable that a periodic solution, if it exists, can not include any part of the line  $i = 0$  because this line is invariant.

Applying Dulac's Test to the system (10) & (11) with the specified choice for  $\rho$  we obtain

$$\frac{\partial(\rho f)}{\partial s} + \frac{\partial(\rho g)}{\partial i} = -(\beta + p + \mu i^{-1}).$$

This function is strictly negative inside the positive quadrant. Thus no periodic solution can exist that is entirely contained within the positive quadrant. As the positive quadrant is positively invariant there can be no periodic solution only partly contained within it.

**A.3. There is no community of problem drinkers when  $R < 1$**  In this section we show that alcoholism is eliminated from the population if  $R < 1$ . Let

$$z(t) = i(t) + r(t).$$

Then from the non-negativity of the solution components, §§ A.1, we have that

$$z(t) \geq 0.$$

Using equations (7) & (8) we have

$$\begin{aligned}\frac{dz}{dt} &= \beta si - \mu i - \mu r, \\ &\leq (\beta s - \mu) i \quad (\text{as } r \geq 0), \\ &\leq (\beta - \mu) i \quad (\text{as } s \leq 1), \\ &= \frac{1}{\mu} (R - 1) i.\end{aligned}$$

Hence

$$z - z_0 \leq \frac{1}{\mu} (R - 1) \int_0^t i d\tau.$$

As  $z(t) \geq 0$  and  $z_0 \leq 1$  we have

$$-1 \leq \frac{1}{\mu} (R - 1) \int_0^t i d\tau.$$

It follows that if  $0 < R < 1$  then

$$\lim_{t \rightarrow \infty} i(t) = 0.$$

### B. When is the endemic steady-state physically meaningful?

In this section we determine when the roots of the quadratic equation

$$G(\hat{i}) = \hat{i}^2 - B\hat{i} + C = 0, \quad (31)$$

are physically meaningful ( $\hat{i} > 0$ ) where the coefficients are

$$B = \frac{R - 1}{R} - \frac{1}{R_0} \frac{\beta}{p}, \quad (32)$$

$$C = \frac{\mu}{p} \left( \frac{1 - R_0}{R_0} \right). \quad (33)$$

The parameters  $R_0$  and  $R$  are defined by equations (13) & (14) respectively. The roots of (31) are given by

$$\hat{i} = \frac{B \pm \sqrt{B^2 - 4C}}{2}, \quad (34)$$

Note that when  $R_0 < R \leq 1$  we have  $B < 0$  and  $C > 0$ . In this case the roots of equation (34) are not physically meaningful because they are either complex ( $B^2 - 4C < 0$ ) or strictly negative ( $B^2 - 4C \geq 0$ ).

Thus we may assume that  $R > 1$ . We consider in turn the cases:  $R_0 > 1$ ,  $R_0 = 1$  and  $R_0 < 1$ .

**B.1. Case 1:  $R_0 > 1$  ( $\beta > \mu + \gamma$ )** In this case the coefficient  $C$  is negative. This means that the discriminant of equation (34) ( $B^2 - 4C$ ) is positive. Thus equation (34) must have one positive root and one negative root. Hence when  $R_0 > 1$  the (physically meaningful) endemic state is single valued.

**B.2. Case 2:  $R_0 = 1$  ( $\beta = \mu + \gamma$ )** In this case the coefficient  $C$  is zero and the roots of equation (31) are  $\hat{i} = 0$  and  $\hat{i} = B$ . When  $R_0 = 1$  the coefficient  $B$  can be rewritten as

$$B = \frac{\mu}{\beta p} \cdot (R_0 - 1) \cdot \left( p - \frac{\beta^2}{\beta - \mu} \right).$$

Recall that

$$R = \frac{\beta}{\mu} > 1$$

and let

$$p_{\text{cr}} = \frac{\beta^2}{\beta - \mu} = \frac{R}{R - 1} \cdot \beta.$$

If  $p > p_{\text{cr}}$  then the non-zero root to equation (18) is positive whereas if  $p < p_{\text{cr}}$  then the non-zero root to equation (18) is negative. Thus when  $R_0 = 1$  the physically meaningful endemic state, if it exists, is single valued.

**B.3. Case 3:  $R_0 < 1$  ( $\beta < \mu + \gamma$ )** In this case the coefficient  $C$  is positive. Thus if  $B < 0$  then the roots of equation (31) are either complex ( $B^2 - 4C < 0$ ) or negative ( $B^2 - 4C > 0$ ). If  $B = 0$  then the roots are purely complex. Thus a necessary condition for positive values is

$$\begin{aligned} B &> 0, \\ \Rightarrow R_0 &> R_{0,\text{cr}} = \frac{\beta}{p} \cdot \frac{R}{R - 1} \quad (\text{assuming } R > 1). \end{aligned} \quad (35)$$

By assumption we have  $R_0 < 1$ . Thus from equation (35) a necessary condition for positive values is

$$p > \frac{R}{R - 1} \cdot \beta = p_{\text{cr}}. \quad (36)$$

We deduce from this equation that there can never be positive solutions when  $\beta \geq p$ .

When both the coefficient  $B$  and the discriminant of equation (31) ( $\Delta_i$ ) are positive then the endemic equation has two positive solutions. The discriminant of

equation (31) can be written

$$\Delta_i = \frac{1}{R_0^2} \cdot G_2(R_0), \quad (37)$$

$$G_2(R_0) = aR_0^2 + bR_0 + c, \quad (38)$$

$$a = \left( \frac{R-1}{R} \right)^2 + \frac{4\mu}{p},$$

$$b = -\frac{2}{p} \left[ \left( \frac{R-1}{R} \right) \cdot \beta + 2\mu \right] < 0,$$

$$c = \frac{\beta^2}{p^2}.$$

The discriminant of the quadratic equation  $G_2$  in (37) is

$$\Delta_{R_0} = \frac{16\mu}{p^3} (p - \beta).$$

From inequality (36) the necessary condition for positivity is  $p > \beta$ . Thus the quantity  $\Delta_{R_0}$  is positive. It follows that the discriminant of equation (31), equation (37), is positive when either

$$0 < R_0 < R_- \quad (39)$$

or

$$R_+ < R_0 < 1 \quad (40)$$

where

$$R_+ = \frac{-b + \sqrt{b^2 - 4ac}}{2a}, \quad (41)$$

$$R_- = \frac{-b - \sqrt{b^2 - 4ac}}{2a}. \quad (42)$$

We now show that  $R_- < R_{0,\text{cr}}$ , where  $R_{0,\text{cr}}$  is defined by (35). As the necessary condition for the positivity of the solution is  $R > R_{0,\text{cr}}$  this shows that solutions in the region  $0 < R_0 < R_-$  are negative. Consider the quadratic equation (38). We have

$$\begin{aligned} \mathcal{G}_2(R_0 = R_{0,\text{cr}}) &= \frac{4\beta\mu R}{(1-R)^2 p^3} \cdot [\beta R - (R-1)p] \\ &< 0 \quad (\text{using inequality (36)}). \end{aligned}$$

As

$$\mathcal{G}_2(R_0 = 0) = c > 0,$$

it follows that

$$0 < R_- < R_{0,cr}$$

Thus the region (39) corresponds to negative values for the endemic steady-state  $\hat{i}$ .

Consequently the condition for there to be two positive solutions is given by equation (36) and the range of values of  $R_\gamma$  over which this occurs are given by equation (40) & (41).

## References

- [1] R.M. Anderson and R.M. May. *Infectious Diseases of Humans* (Oxford University Press, Oxford, 1991).
- [2] C.P. Bhunu. ‘A mathematical analysis of alcoholism’. *World Journal of Modelling and Simulation* **8** (2012), 1214–134. DOI:doi=10.1.1.374.6247.
- [3] F. Brauer and C. Castillo-Chávez. *Mathematical Models in Population Biology and Epidemiology, Texts in Applied Mathematics*, volume 40 (Springer-Verlag, Berlin, 2001), first edition.
- [4] F. Brauer, P. van den Driessche, and J. Wu, editors. *Mathematical Epidemiology, Lecture notes in Mathematics*, volume 1945 (Springer, 2008).
- [5] B. Buonomo and D. Lacitignola. ‘Modeling peer influence effects on the spread of high-risk alcohol consumption behavior’. *Ricerche di Matematica* **63** (2014), 101–117. DOI:10.1007/s11587-013-0167-3.
- [6] A. Cintrón-Arias, F. Sánchez, X. Wang, C. Castillo-Chavez, D.M. Gorman, and P.J. Gruenewald. ‘The role of nonlinear relapse on contagion amongst drinking communities’. In C. Castillo-Chavez, L.M.A. Bettencourt, and G. Chowell, editors, *Mathematical and Statistical Estimation Approaches in Epidemiology* (Springer, 2009), pages 343–360. DOI:10.1007/978-90-481-2313-1\_14.
- [7] O. Diekmann, J.A.P. Heesterbeek, and T. Britton. *Mathematical Tools for Understanding Infectious Disease Dynamics* (Princeton University Press, Princeton, N.J., 2013).
- [8] H. Dulac. *Points Singulieres des Équations Differentielles, Mém. Sci. Math, Fasc*, volume 61 (Gauthier-Villars, Paris, France, 1934).
- [9] C.J. Eastwood, V. Mahajan, and E. Muller. ‘A nonuniform influence innovation diffusion model of new product acceptance’. *Marketing Science* **2** (1983), 273–295. DOI:10.1287/mksc.2.3.273.
- [10] M.G.M. Gomes, L.J. White, and G.F. Medley. ‘Infection, reinfection, and vaccination under suboptimal immune protection: epidemiological perspectives’. *Journal of Theoretical Biology* **228** (2004), 539–549. dx.doi.org/10.1016/j.jtbi.2004.02.015.
- [11] B. González, E. Huerta-Sánchez, A. Ortiz-Nieves, T. Vázquez-Alvarez, and C. Kribs-Zaleta. ‘Am i too fat? Bulimia as an epidemic’. *Journal of Mathematical Psychology* **47** (2003). doi:10.1016/j.jmp.2003.08.002.
- [12] P. Hagedoorn. *The impact of media campaigns on the spread of alcoholism*. Undergraduate honours thesis, School of Mathematics and Applied Statistics, The University of Wollongong, 2015.
- [13] D. Harrison. ‘Australian alcohol consumption at 50-year low, ABS says’. *The Sydney Morning Herald* May 7.
- [14] A.L. Hill, D.G. Rand, M.A. Nowak, and N.A. Christakis. ‘Infectious disease modeling of social contagion in networks’. *PLOS Computational Biology* **6** (2010), e1000968. doi:10.1371/journal.pcbi.1000968.
- [15] C.S. Hindmarsh, S.C. Jones, and L. Kervin. ‘Effectiveness of alcohol media literacy programmes: a systematic literature review’. *Health Education Research* **30** (2015), 449–465. doi:10.1093/her/cyv015.

- [16] H-F Huo and N-N Song. ‘Global stability for a binge drinking model with two stages’. *Discrete Dynamics in Nature and Society* **2012** (2012), Article ID 829386. DOI:10.1155/2012/829386.
- [17] H-F Huo and C-C. Zhu. ‘Influence of relapse in a giving up smoking model’. *Abstract and Applied Analysis* **2013** (2013), Article ID 525461. <http://dx.doi.org/10.1155/2013/525461>.
- [18] H.F. Huo and Q. Wang. ‘Modelling the influence of awareness programs by media on the drinking dynamics’. *Abstract and Applied Analysis* **2014** (2014), Article Number: 938080. DOI:10.1155/2014/938080.
- [19] J. Ireland. ‘Why Tony Abbott’s skol doesn’t sit quite right’. *The Sydney Morning Herald* April 19.
- [20] L. Jódar, F.J. Santonja, and G. González-Parra. ‘Modeling dynamics of infant obesity in the region of Valencia, Spain’. *Computers & Mathematics with Applications* **56** (2008), 679–689. doi:10.1016/j.camwa.2008.01.011.
- [21] J.D. Jones, S.D. Comer, and H.R. Kranzler. ‘The pharmacogenetics of alcohol use disorder’. *Alcoholism: Clinical and Experimental Research* **39** (2015), 391–402. doi:10.1111/acer.12643.
- [22] D.W. Jordan and P. Smith. *Nonlinear Ordinary Differential Equations*. Oxford Applied Mathematics and Computing Series (Clarendon Press, 1989), second edition.
- [23] C.M. Kribs-Zaleta. ‘Sociological phenomena as multiple nonlinearities: MTBI’s new metaphor for complex human interactions’. *Mathematical Biosciences and Engineering* **10** (2013), 1587—1607. DOI:10.3934/mbe.2013.10.1587.
- [24] J.L. Manthey, A.Y. Aidoo, and K.Y. Ward. ‘Campus drinking: an epidemiological model’. *Journal of Biological Dynamics* **2** (2008), 346–356. DOI:10.1080/17513750801911169.
- [25] A. Mubayi, P.E. Greenwood, C. Castillo-Chávez, P.J. Gruenewald, and D.M. Gorman. ‘The impact of relative residence times on the distribution of heavy drinkers in highly distinct environments’. *Socio-Economic Planning Sciences* **44** (2010), 45–56. doi:10.1016/j.seps.2009.02.002.
- [26] G. Mulone and B. Straughan. ‘Modeling binge drinking’. *International Journal of Biomathematics* **5** (2009a), 1250005. DOI:10.1142/S1793524511001453.
- [27] G. Mulone and B. Straughan. ‘A note on heroin epidemics’. *Mathematical Biosciences* **218** (2009b), 138–141. DOI:10.1016/j.mbs.2009.01.006.
- [28] M.A. Nowak and R.M. May. *Virus Dynamics* (Oxford University Press, Oxford, UK, 2000), first edition.
- [29] L. Perko. *Differential Equations and Dynamical Systems*. Texts in Applied Mathematics 7 (Springer, 1996), second edition.
- [30] M.G. Roberts. ‘The pluses and minuses of  $r_0$ ’. *Journal of the Royal Society Interface* **4** (2007), 949–961. doi:10.1098/rsif.2007.1031.
- [31] F. Sánchez, X. Wang, C. Castillo-Chávez, D.M Gorman, and P.J Gruenewald. ‘Drinking as an epidemic — a simple mathematical model with recovery and relapse’. In K. Witkiewitz and G. Marlatt, editors, *Therapist’s Guide to Evidence-Based Relapse Prevention*, chapter 16 (Academic Press, 2007), pages 353–368. DOI:10.1016/B978-012369429-4/50046-X.
- [32] O. Sharomi and A.B. Gumel. ‘Curtailing smoking dynamics: a mathematical modeling approach’. *Applied Mathematics and Computation* **195** (2008), 475–499. doi:10.1016/j.amc.2007.05.012.
- [33] R. Smith? *Modelling Disease Ecology with Mathematics, Differential Equations & Dynamical Systems*, volume 2 (American Institute of Mathematical Sciences, Springfield, MO, USA, 2008).
- [34] C.E. Walters, B. Straughan, and J.R. Kendal. ‘Modelling alcohol problems: Total recovery’. *Ricerche di Matematica* **62** (2013), 33–53. DOI:10.1007/s11587-012-0138-0.
- [35] E. White and C. Comiskey. ‘Heroin epidemics, treatment and ODE modelling’. *Mathematical Biosciences* **208** (2007), 312–324. doi:10.1016/j.mbs.2006.10.008.